

EFFECTS OF KINETIC CAVITY PREPARATION VS.
CONVENTIONAL HANDPIECE PREPARATION
ON THE HUMAN DENTAL PULP

by

Julie M. Collins

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David R. Avery

Jeffrey A. Dean

Susan L. Zunt

Abdel H. Kafrawy

Brian J. Sanders

Chair of the Committee

Date _____

TABLE OF CONTENTS

Introduction.....	1
Review of Literature	4
Materials and Methods.....	36
Results.....	41
Figures and Tables	44
Discussion.....	62
Summary and Conclusions	68
References.....	71
Abstract.....	79
Curriculum Vitae	

LIST OF ILLUSTRATIONS

FIGURE 1	The KCP 200 kinetic cavity preparation unit	45
FIGURE 2a	Kinetic cavity preparation on an extracted human tooth	46
FIGURE 2b	High-speed handpiece preparation on an extracted human tooth.....	46
FIGURE 3	Normal dental pulp tissue	47
FIGURE 4a	Kinetic cavity preparation with no pulp reaction.....	48
FIGURE 4b	Higher power view of kinetic cavity preparation with normal pulp tissue.....	49
FIGURE 5a	High-speed handpiece preparation with pulpal reaction.....	50
FIGURE 5b	Higher power view of high-speed handpiece preparation with odontoblastic displacement.....	51
FIGURE 5c	Highest power view of high-speed handpiece preparation with odontoblastic displacement.....	52
FIGURE 6a	High-speed handpiece preparation with disturbance in odontoblastic layer	53
FIGURE 6b	Higher power view of high-speed handpiece preparation with disruption in odontoblastic layer	54
FIGURE 7a	Pulp stone.....	55
FIGURE 7b	Higher power view of pulp stone with normal pulp tissue	56
FIGURE 8	Diffuse calcifications of the pulp	57
FIGURE 9	External resorption with reversal and repair	58
TABLE I	Kinetic cavity preparation results	59

TABLE II	High-speed handpiece preparation results	60
TABLE III	Other histologic findings.....	61

INTRODUCTION

One of the challenges facing dentists is to reduce the amount of fear and discomfort felt by the patient, while placing a restoration that is both retentive and non-irritating to the dental pulp. The high-speed dental handpiece, with its noise and the vibration transmitted through the hard tissues of the teeth and skeleton, has often been identified as a significant source of anxiety.¹⁻⁴

In the past, the most commonly used dental materials required that the handpiece be used to create a form that would be retentive for the restoration. Kinetic cavity preparation, available since the 1950s, was not frequently used because the cavity form was not retentive for these dental materials.

Two factors brought about changes in cavity preparation. First, studies that evaluated the factors influencing internal stress and fracture of amalgam proved that sharp line angles are not necessary for a long-lasting restoration.^{5,6} Second, the development of composite resin materials and glass ionomers, and the process of direct bonding to enamel and dentin increased the chemical retention of restorative materials and decreased the importance of mechanical retention to some degree.⁷

The dentist has always been concerned with the pulpal effects of any preparation method and of the restorative materials used. Use of the high-speed handpiece with air-water cooling and light pressure has not resulted in lasting pulp response. An animal study using kinetic cavity preparation with 27- μ m particles at 160 psi has resulted in

fewer pulp effects than the high-speed handpiece.⁸ Use of glass ionomer materials for restorative purposes has shown no detrimental effect on the pulp.⁹

The objective of this study was to test the following hypothesis: kinetic cavity preparation results in significantly fewer pulpal effects than does conventional preparation using the high-speed handpiece.

REVIEW OF LITERATURE

AIR ABRASION

The air abrasion technique for cavity preparation, today also referred to as kinetic cavity preparation, has been available to dentists since 1945. In 1943, investigation was begun to find a non-mechanical method that would rapidly cut tooth structure with little or no trauma or discomfort felt by the patient.¹⁰ First described by Robert Black in 1945 as the "airbrasive" technique,¹ this approach to tooth preparation is based on a fundamentally different principle. Rotary instruments rely on mechanical means to remove tooth structure; air abrasion uses kinetics for its action.^{1,2} The mechanical energy from the motor is converted to kinetic energy as abrasive particles pass through the nozzle.¹ These particles are charged with sufficient energy to achieve the cutting action.^{1,4}

The requirements of the system were to use an inexpensive, free-flowing, abrasive agent that could rapidly and efficiently cut tooth structure and that was carried by a non-toxic gas. Aluminum oxide was chosen for its properties of being chemically stable, non-toxic, inexpensive, free-flowing, easily obtainable, and neutral in color.¹ Compressed air, nitrogen, and carbon dioxide were considered for the carrier; compressed air was found to be most nearly ideal;¹ however, the early units used carbon dioxide, which was free of moisture, non-toxic in low concentrations, inexpensive, convenient, and almost universally available.² The first instruments used aluminum oxide particles 30 μm in size

at pressures of 80 psi. Today's air abrasion units provide a choice between 27 and 50 μm particles at pressures of 80 or 160 psi, with compressed air as the propellant of choice.

Another design problem was selection of the proper metal for the nozzle. At speeds of abrasive particles variously reported from 1000 to 1900 ft/sec,^{1-4,10,11} the nozzle would wear within one use of the technique. Sintered tungsten carbide was used because it withstood wear.^{2,11} The internal diameter of the nozzle of 0.018 in was the size able to produce the required velocity.¹¹

Early studies showed several advantages for the patient and the dentist with the use of air abrasion. The foremost advantage to the patient was the lack of pain noted when preparation was done without use of local anesthesia. Goldberg (1952) studied the perception of pain by 1,141 patients and found no pain to be noted by 50.3 percent, mild pain by 40.6 percent, and severe pain by 9.3 percent.¹² If present, pain was described as similar to cold air striking the tooth.⁴ Reasons for pain were stimulation of the pulp due to hyperemia, or a low pain threshold.¹⁰ Goldberg's results showed that 92.3 percent preferred the air abrasive technique to the handpiece preparations used at that time. In general, patient response was overwhelmingly in favor of the air abrasion technique.^{3,4,13}

The sources of pain or discomfort during cavity preparation are pressure, heat, vibration through the bone, and unpleasant sounds.^{1-4,11} With air abrasion, pressure that could be perceived by the patient is eliminated.¹ The abrasive particles work at speeds unobtainable by mechanically driven burs. Pressure is required for cutting with burs due to the slower speed at which they operate.¹ The amount of heat generated is greatly decreased with air abrasion, lacking in pressure. The impact of the particles on the tooth causes the air to rapidly expand, dissipating any heat produced.¹ The sense of vibration is

lost, because the air abrasion particles are too small to create sufficient impact on the tooth. Vibrations caused by the rotary instrument are interpreted as noise through bone conduction,² which is unpleasant to most people.³ This type of noise is eliminated with air abrasion. The whine of the handpiece is also not present.

However, Epstein¹⁴ recommended that even with air abrasion, the factor of patient discomfort should not be minimized. The frictional force of abrasive particles and the decrease in surface temperature of the tooth may be uncomfortable. Local anesthesia was likely to be necessary for other parts of the treatment, such as placement of the rubber dam clamp, and the adjunctive use of rotary or hand instruments.

Mann¹¹ stated that the most important benefit to both the dentist and the patient was that cavity preparation by air abrasion should exert fewer pulpal influences than other methods. The result is fewer instances of hyperemic pulpalgia and less pulpal degeneration following normal operative procedures.

The dentist also benefits through the use of the air abrasive technique. Because the needs for finger bracing and holding the patient's head are eliminated, the amount of fatigue and tension are decreased.¹ The finger bracing needed to aid in the application of force with the rotary instrument can be replaced with a finger rest and a pen grasp with air abrasion. However, because air abrasion cuts rapidly, the dentist's vision must be trained on the tooth structure being removed.¹⁵ The necessary manipulation is dictated by vision rather than the tactile senses.¹⁵ Epstein¹⁴ noted that while the physical fatigue may be reduced, eye fatigue is likely to increase, because vision is the only guiding control.

Another concern with air abrasion relates to soft tissue trauma from the abrasive particles. Studies have shown that there is no demonstrable damage to the soft tissue, even with the particles aimed directly at this tissue.^{1,8,10}

The potential for respiratory effects from abrasive particles was considered. Aluminum oxide is inert and non-toxic. A particle size of 28 μm , which is considered average, is well above the size able to reach the alveoli,³ and particles of this size are effectively filtered by the human breathing apparatus.⁴ Fullmer and Eastman¹⁶ and Kerr¹⁷ studied the effects of aluminum oxide on the rabbit lung and found that respiratory irritation could be produced following inhalation of extremely large quantities of the abrasive. There has been no evidence that inhalation of small amounts, which could be seen with use of the air abrasion technique, would be a serious health hazard to the dentist or patient.^{4,16} No adverse effect on health was reported after the first six years that the technique was used.⁴ With current air abrasion units, particle flow is controlled with better metering and evacuation systems,⁷ which decrease the respiratory risks.

These early studies concluded that the action of gas-propelled abrasives was suited to dental needs. The action obtained is specific for cutting hard materials. It is effective for penetrating and excavating enamel and dentin. The resulting tooth preparation, when hand instruments are used in conjunction with air abrasion, is no different than preparation with rotary instruments.¹⁰

Preliminary reports also noted that while patients generally preferred the air abrasion when it was offered, cost was the main reason that the dentists did not purchase the unit.¹³ Other reasons were the time needed to become proficient with the instrument, the size of the unit, and the abrasion of glass mirrors.¹⁰ Air abrasion may have been

avoided, because it was expected to replace rotary preparation in all cases. There is no reason to expect that any new instrument will be able to perform all duties in operative dentistry.⁴ Air abrasion is best suited for non-traumatic rapid bulk cutting of tooth structure.¹⁰

By the late 1950s, the use of air abrasion had been overshadowed by the Borden air rotor, the first high-speed handpiece.^{7,10} This was attributed to several drawbacks of air abrasion: (1) the standard materials of the day, alloy and gold, required use of rotary instrumentation to achieve the correct preparation; (2) the walls produced with air abrasion were uneven, so that hand instrumentation was needed; (3) air abrasion could not easily remove metal restorations; and (4) the high-volume evacuation available at the time was not sufficient to remove the large amount of abrasive powder.⁷

Air abrasive technology has recently enjoyed a resurgence in interest because of the emergence of resins as a restorative material.⁷ Bonded resins do not require precise outline form. The action of air abrasion produces an etched surface on enamel and dentin, so that the cavosurface is ideally finished for resin placement.¹⁹ The particle flow appears to anneal the surface of the dentin, which closes tubules and creates a superior surface for bonding.¹⁹ Bond strengths that are twice as strong as those using current bonding techniques have been reported.¹⁹

Air abrasion provides a faster and better method of modifying the surface of existing resins or defective sealants that require bonding.⁷ Air abrasion can be used to repair margins of porcelain veneers that are bonded to the tooth with resin.⁷ Use of a handpiece risks a compromise of the bond and formation of microcracks in the porcelain.⁷

With the increased interest in enamel conservation, air abrasion is useful as a preventive technique. The narrowly focused particle stream abrades tooth structure in proportion to particle size, air pressure, and nozzle distance.¹⁸ This provides a conservative means for early interception of pit and fissure lesions. Dark areas that may be early carious lesions or merely stained grooves can be cleaned out with negligible widening between the walls.^{7,18} Early caries may be restored with resin; otherwise, the surface is prepared for placement of sealant.

Goldstein and Parkins⁷ found that, in addition to minimizing heat, vibration, and bone-conducted noise, another aspect of patient discomfort that is not apparent with air abrasion is the odor. They also stated that the presence of an excessive amount of particles has been mitigated with better metering and evacuation systems to control the amount of powder flow.

Given the current bonded resin materials, air abrasion could become a standard method of preparation to achieve comfortable, conserving, and interceptive treatment with improved cavity design.⁷ The question that remains is the degree of resistance that dentists will have against the use of this technology in view of their resistance to the high-speed handpiece at first, fearing injury to surrounding teeth and tissues with use of the 13,000 rpm, compared with the standard of 4,500 rpm.²⁰

HISTOLOGY AND HISTOPATHOLOGY OF THE PULP

To evaluate changes in the pulp, knowledge of the basic histology of the normal pulp is necessary. For a complete review, see Ten Cate.²¹ Dentin and pulp are embryologically, histologically and functionally the same tissue and are considered

together. Histologically, the dentin is permeated by tubular structures that follow an S-shape from the dentinoenamel junction to the pulp. Within the tubules are tissue fluid and odontoblastic processes. Three types of dentin are known: primary, which forms most of the tooth as the tooth is developing; secondary, which forms after root formation is complete and represents the continuing deposition of dentin as the tooth ages; and tertiary, which forms in response to noxious stimuli, only by the odontoblasts directly affected by the stimulus. Tertiary dentin is also known as reparative, reactive, or irregular secondary dentin.

Predentin, the layer between the dentin and the pulp, is unmineralized dentin matrix and is important in maintaining the integrity of dentin. Predentin is thicker in areas where dentin is being formed.

The dental pulp is the soft connective tissue that supports the dentin. Pulp can be divided into four zones, moving from pulp periphery toward the central pulp: the odontoblastic zone, the cell-free zone, also called the zone of Weil or Weil's basal layer, the cell-rich zone, and the pulp core, which contains the major blood vessels and nerves.

There are five cells of the pulp. The odontoblast is the most easily recognized, forming a single layer at the periphery of the pulp with a process extending through the predentin into the dentinal tubules. The fibroblast is the cell occurring in greatest number, whose function is to form and maintain the pulp matrix, and to ingest and degrade collagen. The undifferentiated mesenchymal cells are able to give rise to fibroblasts and odontoblasts, depending on the stimulus. The macrophage is involved in the elimination of dead cells and also participates in the inflammatory response by

removing bacteria and interacting with other inflammatory cells. The lymphocytes are T lymphocytes, which are associated with the immune defense system.

The blood vessels and nerves form a neurovascular bundle, entering the pulp through the apex and accessory canals. They are large in the central core of the pulp and branch peripherally.

With trauma such as cavity preparation, the tooth responds when the odontoblastic process is involved. Reparative dentin will form beneath the damaged dentinal tubules. With more intense injury, an inflammatory response in the pulp will develop, with invasion of neutrophils within a few hours of injury, reaching a maximum at 24 hours. The function of these cells is to control bacterial invasion and infection. If no infection is present, lack of neutrophils does not affect the repair process. After 48 hours, macrophages enter the area. They remove foreign and damaged materials and secrete mitogens for fibroblasts. From undifferentiated perivascular cells are formed odontoblasts, which eventually lay down collagen that becomes mineralized to form reparative dentin. Changes in the microvasculature also occur, with vasodilation and engorgement of blood vessels. Hemorrhage into the area may also occur if the injury is more intense.

Various studies of histologic changes in the pulp have been done for many years. In 1937, Gurley and van Huysen²² described the changes seen following preparation of dog teeth. The histology was evaluated on the basis of the amount of dentin on the pulp side, the infiltration of leukocytes, the amount of blood cells seen in the blood vessels, and the uniformity of the odontogenetic layer. In 1939, Van Huysen and Gurley²³ again conducted a histological study. This time their concern was the amount of cellular

infiltrate at the pulp periphery or deeper, disruption of the odontoblastic layer, and the difference in amount of dentinoid substance. In 1953, Van Huysen and Boyd²⁴ determined that pulpal injury was indicated by the presence of leukocytic infiltration.

It was not until 1952 that the attempt was made to establish set criteria for the assessment of pulp response. This was done to permit more accurate comparative results. Kramer and McLean²⁵ prepared cavities in human teeth, placed several restorative materials, and extracted the teeth at 4 to 58 days. They tried not only to specify the criteria to judge pulpal damage but to indicate some measure of the degree or severity of the reaction. The following were evaluated:

(1) Vascular changes. Severing the apex affected the vessel contents, so that presence of blood vessel congestion was not reliable. Red blood cell extravasation usually accompanied polymorphonuclear infiltration and represented part of the acute inflammatory reaction. The polymorphonuclear infiltration was considered a more reliable assessment of the pulp damage, because hemorrhage may also be seen in the pulp of an extracted healthy tooth.

(2) Protein exudation. The process of eosinophilic protein diffusion in the ground substance, thought to be derived from exudation of blood plasma through the capillary walls, was sometimes seen. Because it was not possible to accept this as causally related in every case, protein exudation was not used as a criterion.

(3) Cellular infiltration. Polymorphonuclear infiltration in the odontoblast layer and focal or diffuse infiltration of inflammatory cells in the pulp tissue other than the odontoblast layer were consistent findings.

(4) Structural changes in the odontoblasts. In some cases, hydropic degeneration of odontoblasts was present, represented by the appearance of vacuoles in the cytoplasm usually between the nucleus and the dentin. Because similar changes were also found in other areas than the preparation site, structural changes in the odontoblasts were not accepted as a criterion.

(5) Secondary dentin formation. This was seen in the area confined to the tubules affected in the preparation. The dentin was normal in some cases and loosely formed in others. This was a consistent finding.

(6) Aspiration of odontoblasts. The partial or complete disappearance of the odontoblast at the pulpal end of the prepared tubule was a striking feature. The odontoblast could be found at various distances within the dentinal tubule.

The main criteria accepted as suitable quantitative indicators of pulpal disturbance and response were: (1) polymorphonuclear infiltration of the odontoblast layer, (2) cellular infiltration of the rest of the pulp, (3) secondary dentin formation, and (4) aspiration of odontoblasts into the dentinal tubules damaged during cavity preparation.

James et al.²⁶ in 1954 also tried to develop more objective criteria for assessment of pulpal responses. Using human teeth, the effects of baseplate gutta percha and preparation with the rotary bur or air abrasion were evaluated. No difference was noted between the preparation methods, so that they were grouped together. Characteristic experimental changes were: (1) a decrease in the number of mature odontoblasts, (2) a break in the continuity of the pulpodentinal membrane, in which the predentin was jagged, with irregular projections, (3) cellular invasion in the layer of Weil (the cell-free zone). The invading cells were lymphocytes and eosinophilic leukocytes, but also

fibroblasts and pre-odontoblasts. This indicated that the reparative process was taking place at the same time as inflammatory changes.

Swerdlow and Stanley²⁷ evaluated the histologic pulpal reaction to cavity preparation through the presence and amount of inflammatory exudate, the absence of intrapulpal abscesses and nodular formations in secondary dentin, the odontoblastic displacement, the decrease in the height of odontoblasts beneath cut tubules, and the presence of vacuolization in the odontoblasts.

Stanley and Swerdlow²⁸ in another study used the presence of cellular exudate, lesions of delayed healing and abscess formation, formation of irregular secondary dentin, cellular displacement, and burn lesions as characteristics for histologic evaluation of pulpal damage.

It is clear that a standardized set of criteria was necessary to permit comparison of similar studies by the same or different people. In 1970, Stanley²⁹⁻³¹ proposed the following characteristics to assess the intensity of responses. These characteristics were based on 13 years of clinical experience involving 45 clinical pulp investigations on 5,500 intact human teeth. These characteristics were:

- (1) cellular displacement into dentinal tubules;
- (2) inflammatory infiltrate into the superficial tissues (the odontoblastic layer, the zone of Weil, and the cell-rich zone) and into the deeper tissues; and
- (3) the presence of a predominant type of inflammatory cell: polymorphonuclear leukocyte, lymphocyte, eosinophil, monocyte, and plasma cell.

Also recommended was examination for special pathologic characteristics: (1) abscess formation, (2) foci of necrosis, (3) lesions of delayed healing, (4) regeneration of odontoblasts, and (5) formation of reparative dentin.

In 1972, the American Dental Association³² published an article that proposed the criteria to determine the harmful effects on the pulp. They included:

- (1) the number and intensity of acute and mononuclear inflammatory cells in the superficial and deeper tissues of the pulp;
- (2) the number of cells displaced into the dentinal tubules;
- (3) capillaries, congested with blood, confined to the odontoblast layer associated with the cut dentinal tubules;
- (4) hemorrhage;
- (5) abscess formation;
- (6) incidence and quantity of irregular (reparative) dentin adjacent to pulpal ends of cut dentinal tubules.

These were accepted in 1979 by the American Dental Association.³³

In 1980, the Federation Dentaire International published their recommended standard practices.³⁴ Specifics for pulpal evaluation were the presence of necrosis, periapical inflammation, inflammation, the degree of inflammation, the predominant inflammatory cells, the presence of a calcified bridge formation, the presence or absence of bacteria, and the type of necrosis.

With these guidelines, investigators may differ in the criteria used in their particular project. The main criteria commonly employed are the reduction in number

and displacement of odontoblastic nuclei into tubules, the presence of inflammatory cells in odontoblastic layer or deeper, and the presence of reparative dentin.³⁵⁻³⁹

The Federation Dentaire International³⁴ also defined categories for inflammation:

(1) Mild – a scattering of inflammatory cells, predominantly chronic inflammatory cells, with identifiable biological characteristics of the residual pulp.

(2) Moderate – focal accumulations of inflammatory cells but no tissue necrosis.

A disruption in the histologic characteristics of the residual pulp may be present.

(3) Severe – total replacement of the residual pulp with inflammatory tissue.

Also to be noted are the extent of tissue necrosis and type of necrosis (liquefaction or coagulation).

PULPAL EFFECTS OF CAVITY PREPARATION

The effects of cavity preparation have been evaluated for many years. The first studies were concerned with temperature rise in the teeth. As rotational speeds of the handpiece increased, the effect on the pulpal tissue began to be studied. The need for coolant on the bur became an issue for safe use. The amount of heat generated, as well as the amount of pressure on the handpiece, were found to have an effect on pulpal response.

In 1951, Vaughn and Peyton⁴⁰ used extracted non-carious molar teeth attached at the dentin-enamel junction to a thermocouple to record the temperature changes with the use of tungsten carbide and steel fissure burs at rotating speeds of 1,310 to 4,000 rpm. They found that near the cutting instrument, the maximum increase in temperature rise occurred within 10 seconds after the operative procedure began. Temperature increases

of 160 °F were noted. Tungsten carbide burs caused less temperature increase than steel burs, and smaller burs less than larger burs. They concluded that both an increase in pressure and an increase in speed had a pronounced effect on a greater temperature rise when using all cutting methods.

Using dog teeth, Lisanti and Zander⁴¹ studied the effect on the pulp with temperature increases of up to 600 °F. Histologic studies were done after the teeth were extracted at intervals of 4, 24, or 48 hours, 1 week, 1 month, and 2 months following preparation. The thickness of dentin varied from 0.81 to 1.52 mm. The changes in the pulp varied directly with the amount of heat generated, ranging from edema and separation of the odontoblast layer to formation of large vacuoles. However, within 2 months, all lesions had healed. The different periods of time between preparation and extraction resulted in observation of several stages of recovery: (1) the area of injury was walled off; (2) necrotic tissue was removed; (3) damaged tissue was replaced; and (4) irregular dentin was deposited opposite damaged dentinal tubules. These results supported their theory that dentin acts as an effective insulating medium due to its low thermal conductivity.⁴² They concluded that the normal dental pulp appears to withstand harmful effects of increases in temperature caused by normal operative procedures.⁴¹

Peyton and Henry⁴³ observed that the heat generated by a rotating instrument is not only be a source of the patient's pain, but can cause pathologic damage to hard and soft tissues. The use of varying speeds of up to 10,000 rpm, with and without coolant, was evaluated. Without use of coolant, a rotating instrument should be used at a maximum of 4,000 rpm with a force of one pound or less in order to keep the temperature rise to 40 °F or less. A temperature rise of over 100 °F was noted in the operating area

when 7,500 rpm and no coolant were used. With coolant, consisting of either air jet or water spray, and with water spray found to be more effective, speeds of 10,000 rpm and force of 1.5 lbs were safe. Also noted was that as the speed increased by 2,000 rpm, the amount of tooth structure removed was doubled, and that high speed (8,000 rpm) operation was effective but hazardous unless carefully controlled.

Another study by Peyton and Henry⁴⁴ found that the choice of instrument, speed of rotation, and use of air or water coolant were factors that controlled temperature rise. With handpiece speeds of 2,000-10,000 rpm, and forces of ½ to 1 ½ lbs, the use of no coolant was safe only at 2,000 rpm, and the use of water coolant was most effective at preventing unacceptable rises in temperature when 10,000 rpm were used. Forces of less than ½ lb were effective due to the increase in efficiency when cutting at high speeds.

As handpiece speed increased, the evaluation of temperature increase continued. Peyton⁴⁵ reported on the effect of air or water spray as coolant and on the effect of ½ to 1 ½ lbs of force using diamond, carbide, or steel burs. Coolant was essential at speeds of 10,000 rpm or more. Water spray was more effective than air in keeping the temperature rise to less than 20 °F. One-half pound of pressure was the most favorable operating condition at 10,000 rpm. Diamond burs caused the lowest temperature rise, and carbide caused lower rises than steel burs.

Peyton⁴⁶ evaluated the new ball-bearing handpieces and found them to operate in a satisfactory manner at speeds higher than 20,000 rpm without excessive heating. Again, air, water, or water spray were essential at these speeds.

Peyton⁴⁷ then compared the effectiveness of air-water spray with different quantities of water at 30,000 and 170,000 rpm. Water volume of 4.0 cc/min was found

to be as effective as 8.5 cc/min in keeping the temperature rise to a minimum when using 4 oz of pressure. This amount of force was most favorable with speeds of 150,000 to 200,000 rpm. At this speed, water volume should not be reduced.

As instrumentation improved, more detailed histologic studies were conducted. Swerdlow and Stanley⁴⁸ conducted studies using 20,000 rpm with water spray. Use of 20,000 rpm was evaluated, because they believed that although equipment capable of producing speeds of 150,000 rpm were available, most preparations were done in the range of 15,000 to 20,000 rpm. Injury to the pulp was seen in all teeth extracted 24 hours after preparation. With use of air-water spray, inflammatory lesions were limited to regions under cut dentinal tubules. Without coolant, extensive damage or abscess formation was seen. When the floor of the cavity was close to the pulp, less reaction occurred with use of air-water spray. They concluded that the potential for recovery was greater in the air-water spray group. They also stated that the cellular displacement in the dentinal tubules was caused by an increase in intrapulpal pressure, which forced cells into the tubules.

Swerdlow and Stanley²⁷ then investigated the high-speed handpiece, operating at 150,000 rpm with air-water spray on a belt-driven contra-angle handpiece. It produced mild traumatic injury to the pulp. The determination of mild injury was influenced by the presence of few infiltrating leukocytes in the early phases and by the absence of intrapulpal abscesses and nodular formation of irregular secondary dentin in later phases.

Marsland and Shovelton⁴⁹ conducted a detailed histologic study based on their belief that damage to the pulp was due to the heat generated during preparation. They supported this determination with the data that pulp injury is not always limited to the

part of the pulp directly beneath the tubules opened during the preparation, that fast and continuous cutting can damage the pulp, and that adequate cooling markedly reduces pulp damage. Human teeth were prepared at speeds of under 5,000 to 15,000 rpm, and the effects were studied after extraction at 28 days post-preparation. Their results indicated that even considerable thicknesses of dentin do not prevent temperature rises of 60 °F in the pulp. However, they did not report on the remaining dentin thickness in their article. The effect of heat was considered likely to disturb normal flow of tissue fluid around the odontoblast and lead to formation of vacuoles, similar to a first-degree sunburn. This accumulation of fluid was probably responsible for the disturbance in the predentin layer. The changes seen were similar to those noted by Lisanti and Zander in dog's teeth.⁴¹ Marsland and Shovelton⁴⁹ stated that smaller temperature changes from slower cutting speeds or shallower preparations may be more limited in effect, causing only coagulative changes in the cytoplasm of odontoblasts. The immediate effect on the pulp, seen consistently, was the aspiration of odontoblastic nuclei into dentinal tubules. This was thought to be a direct consequence of preparation, connected with an increase in temperature caused by the bur. Important conclusions from this study were that use of efficient cooling devices noticeably reduced the thermal damage during cavity preparation and were therefore essential to ensure safe preparation at speeds over 4,000 rpm. Also noted was that pulps showing considerable differences in degree of damage did not cause clinical symptoms, so that lack of pain was not a reliable indicator of a healthy pulp.

Stanley and Swerdlow²⁸ conducted a study of pulp response as related to different preparation techniques. Speeds of 50,000 rpm and above, using either a belt-driven or a

turbine handpiece, with and without coolant, were found to be less traumatic to the pulp than 6,000 and 20,000 rpm handpieces. All teeth with remaining dentin thickness of greater than 2.0 mm were eliminated, because very little to no response was expected with this much dentin. Coolants produced a more favorable response when used at high speeds compared with low speeds. Without coolant, no benefits from intermittent grinding were observed. Preparation time was not important when using higher speeds if adequate coolant was used to control frictional heat. In summary, the use of high speed, controlled temperature and light load resulted in minimal pathologic pulpal alteration.

The pulpal effects of pressure or force used on the handpiece, with the related increase in temperature, were also a concern. Jeserich⁵⁰ reported that forces used by dentists in cavity preparation ranged from 1 lb to 25 lbs. With the use of carbide or diamond burs rotating at speeds over 10,000 rpm, cutting of tooth structure is efficient and effective at forces less than ½ lb.⁴⁴ Peyton and Henry⁴⁴ stated that a slight increase in force caused a greater degree of temperature rise than a corresponding increase in rotational speed. Forces of less than ½ lb are effective with high-speed handpieces. McGehee et al.¹⁵ noted that the pressure exerted by the abrasive stream is approximately 12 to 14 g, or less than ½ oz.

Stanley and Swerdlow⁵¹ evaluated the effects of pressure through observation of the amount of leukocytic infiltration and displacement in the pulp. The incidence and intensity of this inflammatory response was determined. Average remaining dentin thickness was 0.72 to 0.88 mm. Inflammatory response was seen when the force applied to the tooth during preparation exceeded 8 oz, with more severity as the pressure increased. The use of coolant prevented burn lesions in the pulp but did not prevent this

inflammatory response. Continuous or intermittent heavy pressure will produce more damage. These studies evaluated pulpal response at 48 hours after preparation. Previous studies have reported that pulpal changes in response to tooth preparation are reversible, with no lasting damage from low speed, high speed or air abrasion found at 28 days post-preparation.⁵²

With the invention of the air abrasion instrument, comparative studies were undertaken. Lefkowitz et al.⁵² compared the pulp response of new technologies to the standard at that time, the low-speed handpiece that rotated at 5,000 rpm and that was used with intermittent pressure. Preparations were made in sound teeth of dogs and humans, and extractions were done at 1, 7 and 28 days. They compared three methods: (1) the high-speed handpiece, rotating at 24,000 rpm, using continuous water stream; (2) the air abrasion unit; and (3) the ultrasonic unit, which is a rapidly vibrating unit that operates in a field of air, water and abrasive particles similar to those used in the air abrasion unit. Although all remaining dentin thicknesses were not reported, those seen in photomicrographs of ideal preparations ranged from 0.8 to 1.8 mm. This study found no evidence of localized injury to the pulp in relation to any of the preparation methods. The conclusion was that pulp tissue appears to be highly resistant to the necessary insults of tooth preparation for restorative purposes.

Regarding effects of temperature as related to preparation with air abrasive technology, both Peyton and Henry⁴⁴ and McGehee et al.¹⁵ found very little temperature change with air abrasion. The pressure used to mechanically prepare the tooth with the high-speed handpiece generates heat. Air abrasion relies on the kinetic energy of the particles to create the cutting action, so that little or no temperature increase occurs. The

impact of the particles on the tooth causes the air to expand, which dissipates any heat produced.¹ Peyton and Vaughn⁵³ found the average temperature change to be 3.5 °F, from -2.5 °F to +1.0 °F. Peyton and Henry⁴⁴ noted that there may be a 4-to-5-°F increase in the immediate area of cutting, depending on the angle of the instrument and on control of the abrasive material. A cooling action of 3-to-5-°F decrease in temperature was also observed. McGehee et al.¹⁵ reported similar findings: an increase in temperature of only 3.5 °F, and a cooling effect of 1 to 5 °F. They compared this to the increase in temperature related to rotary instrumentation of 140 °F, by using a steel bur rotating at 4,000 rpm.^{15,53}

Laurell et al.⁸ compared the pulp response due to high-speed handpiece preparation with responses to kinetic cavity, or air abrasion, preparation. Preparations were made in dog teeth, and extractions were done at 72 hours. The histologic status of the pulp was evaluated on the basis of cellular displacement, pulpal disruption, and pulpal inflammation, according to the criteria proposed by Stanley.^{29,30} Kinetic cavity preparation was found to result in the same or fewer pulpal changes than preparation with high-speed handpiece and rotary burs.

EFFECTS OF CAVITY DEPTH ON PULP RESPONSE

The response of the pulp to dental materials has long been thought to be related to the depth of the cavity preparation. The distance from the outermost enamel toward the pulp is less important than the thickness of dentin remaining between the restorative material and the pulp. Many studies have evaluated this theory.

In 1939, Van Huysen and Gurley²³ prepared shallow, medium and deep cavities in the teeth of young dogs and left the cavities exposed to oral fluids for 4, 6, or 8 days. Remaining distance from the cavity floor to the odontoblastic layer ranged from 0.85 to 0.325 mm. They concluded that the severity of pulp reaction increased with an increase in cavity depth, but that the type of reaction may be different in every case.

Shroff,⁵⁴ in an investigation of the effects of filling materials and cavity preparation on the human pulp, first defined "effective depth." Effective depth is the distance, or thickness of dentin, between a given point on the cavity floor and the margin of the pulp chamber measured in the direction of the dentinal tubules connecting that point with the pulp. An effective depth of less than 2 mm resulted in injury to odontoblasts in almost all cases. With effective depth of 0.5 mm or less, a severe destructive reaction occurred, from which the pulp was unlikely to recover. With greater than 0.5 mm, the odontoblasts were likely to recover, and dentin was deposited in relation to the number of odontoblasts that survived. With greater than 2 mm effective depth, tubular secondary dentin was formed.

In a study by James and Schour⁵⁵ to evaluate the dentinal and pulpal changes following cavity preparation and filling materials in dogs, the degree of inflammation increased with an increase in cavity depth. The amount of irritation produced could be more accurately evaluated when the preparation was deep.

Swerdlow and Stanley^{27,28} and Stanley^{29,31} reported that the inflammatory response in the pulp was greatest in the area where the preparation was closest to the pulp. They stated that 2.0 mm of dentin thickness between the floor of the cavity

preparation and the pulp will provide an adequate insulating barrier against more traumatic heat-producing operative techniques and most restorative materials.

Because dentinal tubules are S-shaped, Swerdlow and Stanley²⁷ attempted to discover if the actual length of the tubules, rather than the direct distance from the pulp to the preparation floor, would be useful in estimating the pulp response. No improvement in assessing the statistical significance was obtained when using these different measurements.

Seltzer et al.,⁵⁶ in a study to evaluate the effects of pressure, found little or no histologic change in the pulps of dogs and monkeys when the remaining dentin thickness was 1.2 mm or greater. With a thickness of less than 1.2 mm, severity of pulpal change was proportional to depth.

Mitchell et al.⁵⁷ used monkey teeth in an attempt to determine the relation of cavity depth to pulp reaction. Zinc oxide-eugenol and silicate cements were used to restore the preparations, which were evaluated at 7 or 34 days. Pulp responses were mild if the dentin thickness was 0.4 mm or greater. As thickness decreased, the reactions increased from moderate to severe.

Dowden,⁵⁸ in a response to Stanley's statement that dentin thickness over 2.0 mm afforded pulp protection,²⁹ objected. He stated that many of his cases demonstrated a strong pulpal reaction when 2 mm or more of dentin remained. He believed that damage to the cell process and destruction of the protoplasm could cause the cell to degenerate at any distance.

Dickey et al.⁵⁹ evaluated the response of the human tooth to a composite restorative material. Again, floor thickness was assessed and found to be inversely

proportional to pulp response. A more intense reaction was noted with less remaining dentin.

Plant and Anderson⁶⁰ came to a different conclusion. They used a range of restorative materials to investigate the relationship between cavity depth and pulpal response. Calcium hydroxide and phosphoric acid, polycarboxylate, silicate, accelerated zinc oxide-eugenol, zinc phosphate, and zinc oxide-eugenol were placed in cavities prepared to an average depth of 0.751 mm. Only the response to accelerated zinc oxide/eugenol liner was found to be related to the remaining dentin thickness. Statistical analysis indicated that the pulp reaction was not directly related to cavity depth, which was not in agreement with the findings of most previous studies. The results did agree with Dowden.⁵⁸

Plant et al.⁶¹ began to look at bacterial leakage in relation to pulpal response, taking into account the remaining dentin thickness. They found more occurrence and severity of inflammation with closer proximity of bacteria to the pulp, but that the dentin thickness was not a factor. These results were reproduced by Tobias et al.³⁹ in 1991.

Plamondon et al.⁶² investigated the pulp response to the periodontal ligament injection using both deep and shallow preparations in dog teeth. Dentin thickness was not precisely measured, but estimated in the amount of distance from the dentinoenamel junction to the pulp as shallow-moderate, deep, or very deep (near exposure). The periodontal ligament injection did not produce a more severe reaction; only an increase in preparation depth resulted in a more marked response in both experimental and control specimens.

Lee et al.⁶³ used ferret canines to compare the effects of preparation depth and bases on odontoblasts and on the rate of dentin formation. The deepest preparations markedly suppressed or paralyzed and possibly destroyed the odontoblasts. Also associated was the formation of very irregular dentin. Less dentin formation was seen with the deeper preparations through the 40-day observation period. The basing materials had little effect on odontoblast activity or rate of dentin formation.

In general, the effect of depth of cavity preparation continues to be related to amount and intensity of pulpal response. Other factors, such as bacterial penetration into the restoration, add another variable to the subject. Mjor⁶⁴ recommends that only preparations in the inner third of the dentin are acceptable for study of the biologic effects of restorative materials. The Federation Dentaire International³⁴ advocates that for testing restorative materials, the remaining dentin thickness be limited to 1 mm or less.

PULPAL EFFECTS OF GLASS IONOMER CEMENT

In 1972, Wilson and Kent⁶⁵ first reported on a new translucent cement, the glass ionomer or aluminosilicate polyacrylate (ASPA) cement. This new cement, based on the rapid hardening between ion-leachable glass powders and aqueous solutions of polyacrylic acid, was developed for a variety of uses such as restoration of anterior teeth and erosion cavities, general cementation purposes, and cavity linings. Glass ionomer was similar to silicate cement in the powder, but replaced the phosphoric acid component to decrease the irritation associated with it. Studies of the pulpal effects of this material were soon undertaken.

An early study by Klotzer⁶⁶ in 1975 compared pulpal response related to ASPA versus silicate cement in monkeys. Histologic evaluation was completed 28 days after Class V preparations were made. ASPA was found to be irritating, but less so than the silicate cement. However, because of the limited number of teeth involved, only preliminary conclusions could be drawn.

Dahl and Tronstad⁶⁷ also used monkey teeth to evaluate ASPA and silicate cements. The histologic status of the pulp was evaluated 8 days after preparation. The biologic compatibility of the glass ionomer was superior to that of silicate cement.

In 1978, Tobias et al.³⁵ compared the effects of filling material and luting material glass ionomer cements to each other and to zinc oxide-eugenol and Kalzinol, a bacteriocidal cement. The powder-to-liquid ratio of glass ionomer filling material is 3:1, while that of luting material is 2:1. Both human and ferret teeth were used. Evaluation was done on human teeth at 1 to 224 days, and on ferret teeth at 24 hours, 1 week, 2 weeks, 4 weeks, and 1 year. Teeth with bacteria found in the tubules were excluded. Results indicated that ASPA filling material was more irritating to the pulp than were zinc oxide-eugenol or Kalzinol, but that the changes were not marked and resolved by 28 days. The residual dentin thicknesses in experimental and control teeth were similar in humans and ferrets, so that the difference in pulpal response was not thought to be related to cavity depth. Between the two glass ionomers, the luting material was slightly more irritating than the filling material, even with greater dentin thickness under the luting material. These effects were visible only up to 7 days.

Nordenvall et al.⁶⁸ studied the pulp reactions and presence of bacteria under ASPA restorations in acid-etched and unetched cavities compared with those under

composite restorations in human teeth. In the etched/unetched group, histologic evaluation was done after 40 days or 70 to 90 days. Remaining dentin thickness was 0.0 to 0.4 mm. No pulp inflammation was found, even in those teeth with pulp exposures. No bacteria were found. In the unetched ASPA/composite group, evaluation was done after 11 to 68 days, or 101 to 117 days. Remaining dentin thickness was 0.0 to 1.8 mm. Inflammation and bacteria were found in 11 of 19 teeth restored with composite. Bacteria were found in four of 19 teeth restored with ASPA, and these findings were questionable in two of the four. Only the two teeth with definite bacterial presence showed inflammation. The conclusions reached were that the glass ionomer was not irritating to the pulp, and that pulp inflammation is associated with infection of the cavity walls.

Kawahara et al.⁶⁹ compared the biologic properties of glass ionomer cement using the tissue culture method as well as animal teeth. The tissue culture method evaluated the cytotoxic effects on the basis of cell growth and morphological change. The cells showed a weaker reaction to glass ionomer than to zinc oxide-eugenol or to polycarboxylate cement. Monkeys were used for the *in vivo* study. Pulp exposures were made, and the teeth were restored with glass ionomer or zinc oxide-eugenol. Evaluation was done 2 months after preparation. No significant difference in pulp tissue reaction was found.

Cooper⁷⁰ used human teeth to compare the effect of glass ionomer cement mixed as a filling material and an experimental mix of the same material for a luting material. Teeth were extracted 2 to 179 days later. More severe reaction was seen with the luting material. Both glass ionomer cements caused more odontoblast aspiration, changes in the

odontoblast layer, and inflammatory cell infiltration into the odontoblast layer than did the control, zinc oxide-eugenol. These changes occurred more frequently in teeth extracted early in the experimental period. Most had resolved toward the end of the experimental period.

In 1981, Pameijer et al.³⁶ reported on the reaction of the monkey pulp tissue to Chembond, another chemically cured glass ionomer. At 5 days, 1 month, and 3 months, no inflammation, odontoblastic displacement, or reparative dentin formation was seen in 48 preparations. They concluded that Chembond was well-tolerated by the pulp. These results were reproduced by Heys et al.⁷¹ in 1987, in a study on the effects of glass ionomer luting agent.

Paterson and Watts,⁷² also in 1981, found that with direct placement of ASPA on the exposed pulp of rat molar teeth, pulpal necrosis and inflammation of the periapical tissues was a common finding. Histologic evaluation was done at 28 days. This was not seen when calcium hydroxide was used on the exposed pulp. The recommendation was made that calcium hydroxide base should be used in all deep preparations.

Pameijer and Stanley⁷³ compared the use of normal consistency Chembond to the use of a thinner mixture. Primate teeth were prepared to a minimal dentin thickness of 1 mm. Teeth were extracted at 4, 25 and 56 days. Histologic examination revealed that normal consistency glass ionomer resulted in abscess formation compared to zinc phosphate cement when remaining dentin thickness was less than 0.53 mm. Thinner-mixed glass ionomer washed out, leading to carious exposures and pulp lesions. Conclusions were that glass ionomer should be mixed in correct proportion and that a calcium hydroxide liner should be placed in areas where remaining dentin is thin.

Ucok⁷⁴ in 1986 used 60 human teeth to compare ASPA to silicate and zinc oxide-eugenol cements. Preparations were made to a remaining dentin thickness of approximately 1 mm. Teeth were extracted at 3 to 5 days, 27 to 33 days and 85 to 95 days. Histologic examination showed that reaction from all three cements was similar at the short interval. ASPA caused an intermediate response at the 27 to 33 day period. The reaction to silicate cement worsened over time, unlike ASPA, which improved.

Felton et al.⁷⁵ evaluated a light-cured glass ionomer cavity liner in comparison with chemically cured Ketac Bond. Monkey teeth were prepared, and the smear layer was either left intact or removed using 6.0 percent citric acid. A composite resin restoration was placed, and the histologic status evaluated at 8 and 35 days. All teeth showed only minor pulp reaction. The citric acid-treated teeth showed more moderate pulp inflammation than the non-treated teeth. The results demonstrated that light-cured glass ionomer did not impair healing of pulp tissue.

The effect of glass ionomer luting cement, Aquacem, compared with the reaction of other luting cements, zinc phosphate and Poly F, was studied by Plant et al.⁶¹ Occlusal preparations were made in human premolar teeth. Extractions were done at 1 to 10 days, 11 to 30 days and 31+ days. On histologic examination, inflammation was seen with Aquacem and zinc phosphate, but Poly F and the control cement, zinc oxide-eugenol, had only a bland response. A positive correlation was noted between the amount of bacterial leakage and the amount of inflammation. The likelihood and severity of inflammation were increased with closer proximity of bacteria to the pulp, consistent with the view that both the dental material and bacteria are responsible for a pulpal response. The inflammation was not affected by the thickness of remaining dentin.

Paterson and Watts⁷⁶ attempted to separate the causes of pulpal inflammation due to the dental material from that due to bacterial presence. Using germ-free rat molars, preparations were made and the pulp exposed with an explorer. ASPA cement was placed over the pulp. After 28 days, the pulp tissue was fixed and evaluated. Localized pulp necrosis with the inhibition of calcific repair was found. They concluded that linings should be placed in mechanically prepared cavities when using glass ionomer cements.

Dogon et al.³⁷ investigated the pulp response to visible light-cured glass ionomer used as a liner or base. Monkey teeth were used to compare this liner with dentin primer or calcium hydroxide base. All teeth were then restored with composite resin. The animals were sacrificed at 4, 32, and 90 days. There was no significant difference in the histologic status of the pulp with glass ionomer or any other treatment. Reparative dentin was noted in many specimens when the remaining dentin thickness was 0.5 mm or less in all groups at 90 days.

Tobias et al.³⁸ studied the response of anhydrous luting cement with previous results for conventional luting cement. Anhydrous glass ionomers have better physical properties and mixing and handling characteristics. The polyacrylic acid is incorporated into the powder by freeze-drying, and a consistent mix is obtained by adding distilled water from a calibrated dispenser to a measured quantity of powder. In Tobias' study, ferret teeth were used. Histologic examination was done at 7, 14, 28, and 183 days. Results stated that the type of material had no direct association with the degree of inflammation but exerts an indirect influence via antibacterial properties and influence on microleakage.

Gaintantzopoulou⁹ reported on the pulpal effects of light-activated glass ionomer lining cements. Deep preparations were made in dog teeth. The pulp response was studied at 1, 4 and 12 weeks. Microscopic findings indicated that pulpal reactions were minimal and comparable to those produced by chemically set glass ionomer lining cement and zinc oxide-eugenol cement.

In another study by Tobias et al.,³⁹ two seimhydrous glass ionomer luting cements were used to evaluate pulp irritation and association between microbial leakage and pulpal inflammation. Ferret teeth were prepared. A standard luting cement with fluoride was compared to a luting cement with zinc, with a bacteriocidal cement as control. Evaluation was done at 7, 14, 28, and 91 days. The conclusions were that pulp inflammation was related to microleakage within the cavity and not to the thickness of remaining dentin, and that more severe inflammation resulted from bacteria in a location closer to the pulp.

Mjor et al.,⁷⁷ in view of the general agreement that restorative glass ionomer cements were more biocompatible than luting cements, evaluated the pulpal effects of both types. Using dog teeth and evaluating histologic response at 2 or 6 weeks, investigators found only slight reactions. Most inflammatory reactions were due to bacteria at the tooth/filling interface. Bacteria were often localized to a small area on the floor of the cavity with a correspondingly small area of pulp showing an inflammatory reaction.

Felton et al.⁷⁸ conducted a study relating the effect of light-cured glass ionomer cavity liner to chemically-cured Ketac Bond restorative material. Monkey teeth were prepared and evaluated at 7, 14 and 35 days. A remaining dentin thickness of 0.6 +/-

0.18 mm was reported. No difference in pulpal response between the two materials was found. Minimal pulpal reaction was noted, with excellent pulpal responses to both materials. Use of the light-cured material did not impair normal healing response in the pulp with a deep Class V cavity preparation.

Stanley⁷⁹ evaluated newer formulations of glass ionomer cements that have had more acids introduced to reduce setting time and enhance other characteristics. He reported that glass ionomer cements are more irritating, especially when used as luting agents in areas where the remaining dentin thickness is 0.5 mm or less.

In summary, the histologic effects of glass ionomer cement have been studied in great detail for many years. Glass ionomer cements are well-tolerated by the pulp. Instances of inflammatory response were related to bacteria within the dentinal tubules. Overall, this type of cement has been found to be biocompatible and safe for use with the human dental pulp.

MATERIALS AND METHODS

EXPERIMENTAL SPECIMENS

Following approval by the Institutional Review Board, 26 healthy erupted permanent teeth were selected. Seven patients participated in the study, two male and five female. Patient ages ranged from 9 to 15 years old. There was no clinical or radiographic evidence of caries, and no restorations were present. The presence of sealants did not exclude a tooth from the study.

The teeth were obtained from patients who required extractions for orthodontic purposes. These patients were selected, because they were most likely to have virgin teeth with large pulp chambers that would give a reliable response to the experimental methods. A minimum of two teeth per patient was needed to allow for differences in response by different patients.

CAVITY PREPARATIONS AND RESTORATIONS

Class V cavity preparations were made. Thirteen teeth were prepared with the kinetic cavity method, and 13 were prepared with the high-speed handpiece. The teeth were randomly assigned to each preparation group. When two teeth were prepared, a coin toss was used to select the tooth receiving kinetic cavity preparation, and the contralateral tooth was prepared with the high-speed handpiece. When four teeth were used, one maxillary and one mandibular were randomly assigned to the kinetic cavity

preparation group, and the remaining two teeth were prepared with the high-speed handpiece.

Preparation dimensions were as close to 2 mm by 4 mm as possible (Figure 2). All preparations were made by the same operator. For conventional high-speed preparations, a Star high-speed handpiece (Den-Tal-Ez Inc., Valley Forge, PA) rotating at 400,000 rpm with a new 330 bur was used with high volume evacuation. No water coolant was used to try to elicit a greater intensity in pulpal response. For kinetic cavity preparations, the KCP 2000 air abrasion unit (American Dental Technologies, Troy, MI) (Figure 1) was used. Aluminum oxide particles of 27 μm at 160 psi were used, with evacuation by the manufacturer-supplied suction system. For high-speed preparations, the patients were anesthetized with 2.0 percent Lidocaine with 1/100,000 epinephrine (Astra, Westborough, MA), by using approximately 0.9 ml per tooth. One patient required anesthesia for kinetic cavity preparation.

The preparations were restored with glass ionomer restorative material (Photac-Fil, Espe, Seefeld, Germany) per the manufacturer's instructions. The teeth were extracted under local anesthesia 10 to 15 days after the restorative procedure.

HISTOLOGIC PREPARATION

Following extraction, the teeth were immediately placed in 10 percent formalin solution for approximately one week. If the apex of the tooth was closed, the apical one-third of the root was removed to allow penetration of formalin.

Histologic preparation was done according to standard laboratory procedures in the Indiana University Histology Laboratory, as recommended in the Manual of Histologic

Staining Methods.⁸⁰ The teeth were decalcified in 5 percent formic acid. The formic acid was replaced daily until decalcification was complete. Approximately two weeks later, when decalcification was complete, the teeth were processed for routine paraffin embedding. Decalcification was evaluated as follows: every few days, 1 cc of saturated ammonium oxalate solution was added to 5 cc of formic acid solution removed from the specimen bottle. When no precipitate formed, the specimen was considered to be completely decalcified. An overnight bath of running water was used to completely remove the formic acid from the specimens. They were then dehydrated with ethyl alcohol, cleared in xylene, infiltrated with molten paraffin wax for 48 hours, and embedded in paraffin. Serial histologic sections cut 7- μ m thick in the buccolingual plane were made. When the sections were observed to include the preparation, approximately 5 sections of every 25 were saved. Every third section was stained with hematoxylin and eosin for histologic observation. The slides were numerically coded according to preparation method to prevent bias on the part of the examiners.

HISTOLOGIC ASSESSMENT

The slides were examined microscopically. Remaining dentin was measured with a micrometer eyepiece.

Based on the recommendations of the Federation Dentaire Internationale,³⁴ pulp reaction was graded as follows:

No reaction – no demonstrable changes in the pulp.

Mild reaction – odontoblastic displacement within the dentinal tubules, but virtually no pulp inflammation.

Moderate reaction – leukocytic infiltrate in the peripheral portion of the pulp beneath the cut dentinal tubules.

Severe reaction – dense leukocytic infiltrate extending toward the central pulp, with or without microabscess formation.

STATISTICAL ANALYSIS

A sample size of 21 teeth per preparation method was needed to have 80 percent power to detect differences in pulp response at alpha level of 0.05.

The generalized estimating equation method was to be used to compare differences in pulp reaction. When applied to a model for ordinal data, this method can account for correlations that may exist between teeth taken from the same patient.

No statistical analysis was required because the sample size was small, there was little difference in average remaining dentin thickness, and few teeth were noted to show a response to either preparation method.

RESULTS

A total of 26 teeth were prepared, 13 with the kinetic cavity method and 13 with the high-speed handpiece. One tooth per arch was selected for preparation with the kinetic cavity technique; the contralateral tooth was prepared with the high-speed handpiece.

Of the preparations made with the kinetic cavity method (Table I), the depth of the preparation involved only enamel in 4 of 13 specimens. The dentin thickness in these teeth was not measured. Histologic examination was done, and no pulpal reaction was found (Figure 3). In the 9 specimens that included dentin in the preparation, the remaining dentin thickness ranged from 1436 to 2329 μm , with a mean of 1965 μm . One of the specimens showed a pulpal reaction that was classified as mild. Microscopic examination of this specimen demonstrated a decrease in density of the odontoblastic layer, indicating aspiration and degeneration of odontoblasts in the dentinal tubules during the time period between tooth preparation and extraction. The 8 remaining specimens showed no pulpal reaction (Figure 4).

Of the preparations made with the high-speed handpiece (Table II), the dentin was unaffected in 2 of 13 specimens. Again, dentin thickness was not measured, but the specimen was examined for histologic changes. No pulpal reaction was observed in these 2 specimens. The remaining dentin thickness in the 11 specimens with dentin prepared ranged from 1548 to 2720 μm , with a mean of 1930 μm . Three specimens showed pulpal reactions, classified as mild, with aspirated odontoblasts seen in one specimen (Figure 5),

and decreased density of the odontoblastic layer in two specimens (Figure 6). Mild inflammation was noticed in one specimen, and was manifested by leukocyte margination and slight lymphocytic infiltration beneath the cut dentinal tubules. No pulpal reactions were seen in the remaining 8 specimens.

There were other histologic findings of note (Table III). The presence of pulp stones (Figure 7) was also noted in 4 of the 26 specimens, seen in two patients. Three of these teeth had been prepared with the kinetic cavity method, and one with the high-speed handpiece. Diffuse calcification of the radicular pulp (Figure 8) was seen in 4 specimens, all from the same patient. Two teeth were prepared with each of the preparation methods. External root resorption with reversal and partial repair (Figure 9) was noticed in one specimen; this patient had undergone orthodontic treatment prior to extraction.

FIGURES AND TABLES

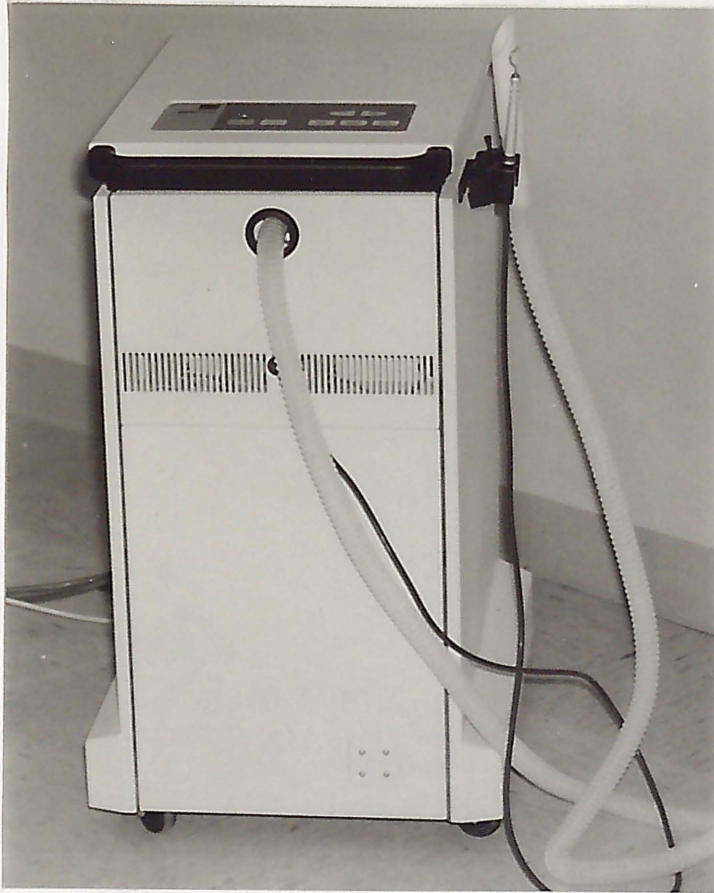


FIGURE 1. The KCP 2000 kinetic cavity preparation unit (American Dental Technologies, Troy, MI).

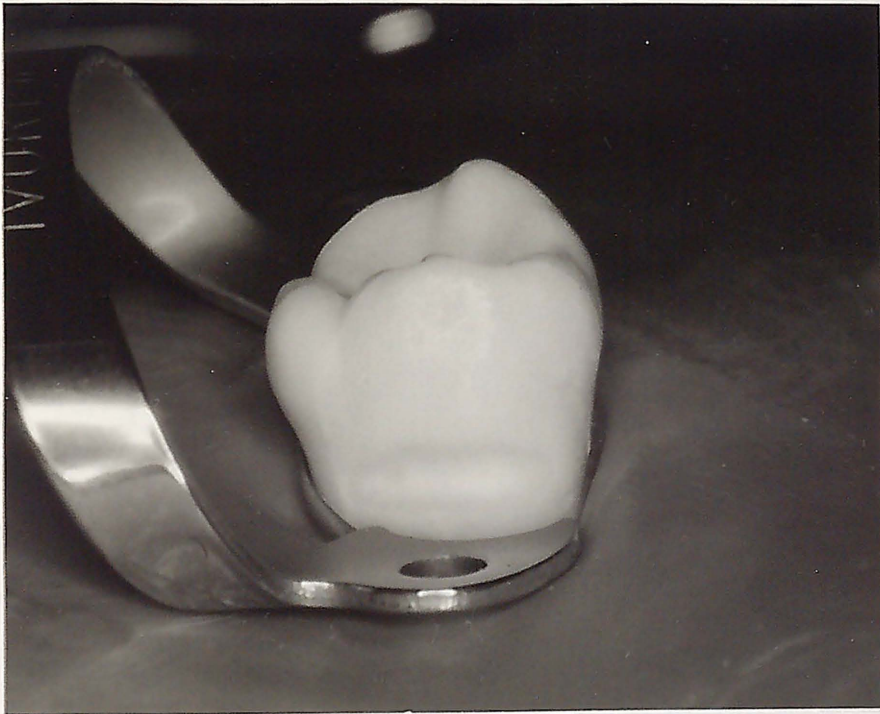


FIGURE 2a. Cavity prepared using the kinetic cavity technique on an extracted human tooth.



FIGURE 2b. Cavity prepared using the high-speed handpiece and 330 bur on an extracted human tooth.



FIGURE 3. Normal dental pulp tissue. The predentin layer is of uniform thickness. The odontoblastic layer is intact with no evidence of aspiration into dentinal tubules. The blood vessels are not engorged. No leukocytic infiltrate is noted. Specimen was prepared with the high-speed handpiece. (Hematoxylin and eosin stain.)

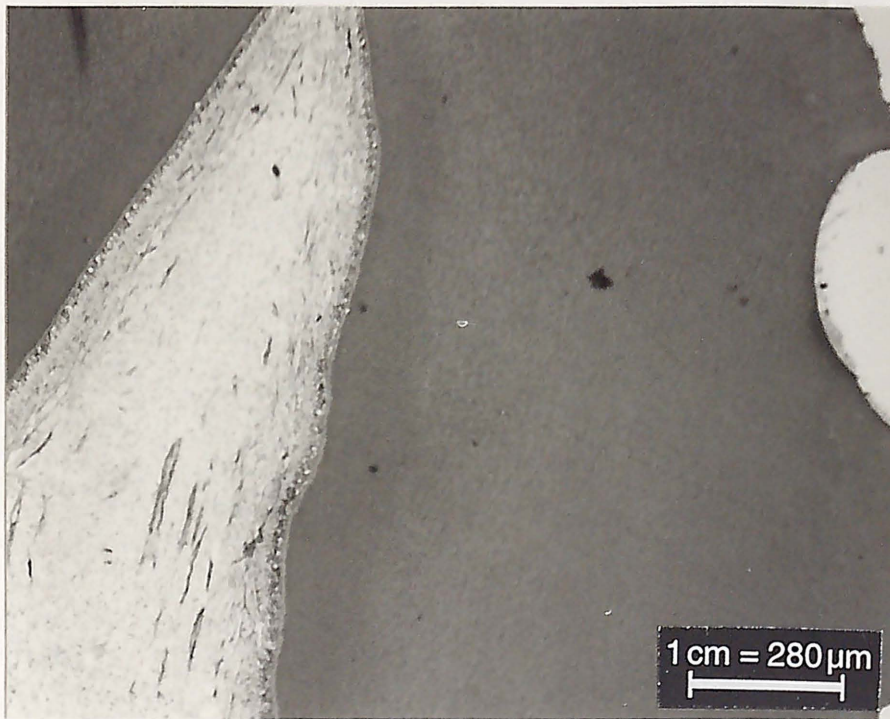


FIGURE 4a. Kinetic cavity preparation. No pulp reaction is seen. (Hematoxylin and eosin stain.)

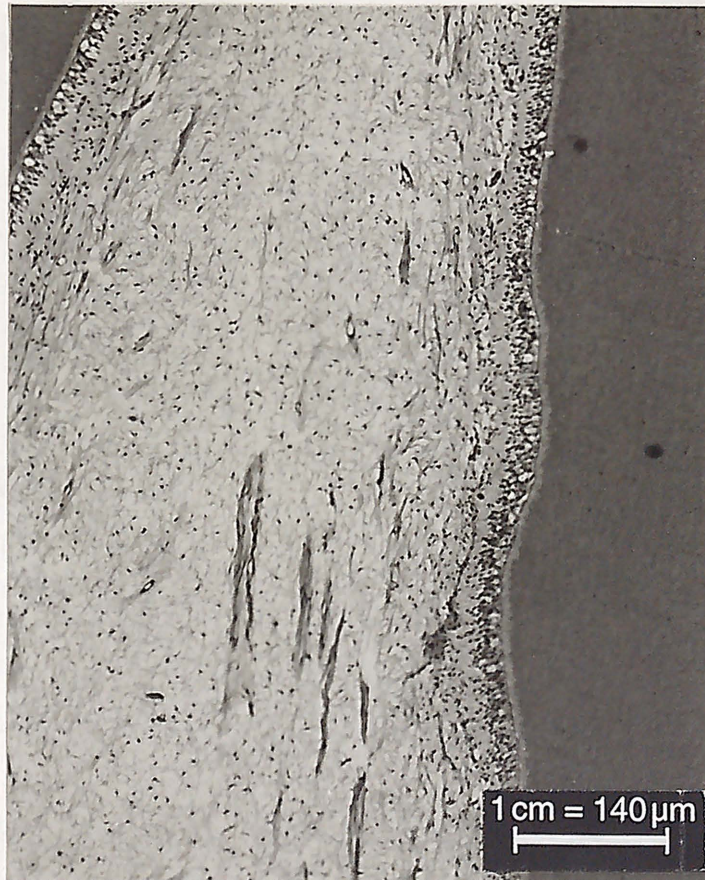


FIGURE 4b. Higher magnification of Figure 4a. The predentin and odontoblastic layers are normal. No engorgement of blood vessels or leukocytic infiltrate is seen. (Hematoxylin and eosin stain.)

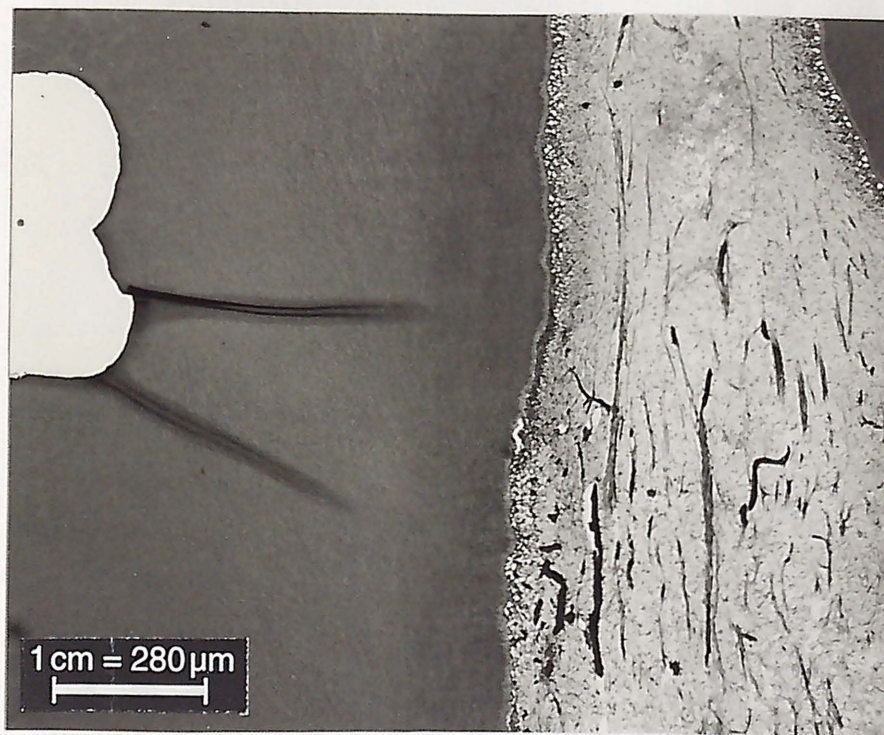


FIGURE 5a. High-speed handpiece preparation. The odontoblastic layer is disrupted. The blood vessels are engorged. The dark-staining folds in the dentin are artifact. (Hematoxylin and eosin stain.)



FIGURE 5b. Higher magnification of Figure 5a.
Displacement of odontoblastic cell bodies into
the dentinal tubules and engorgement of
blood vessels are evident. (Hematoxylin and
eosin stain.)

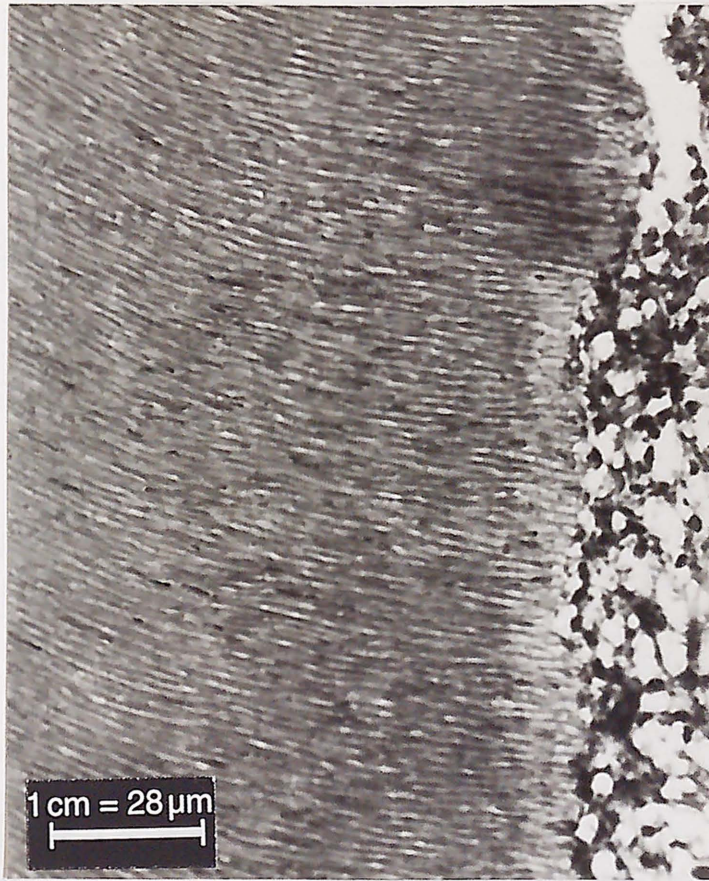


FIGURE 5c. Higher magnification of Figure 5b showing displacement of odontoblastic cell bodies into the dentinal tubules. (Hematoxylin and eosin stain.)

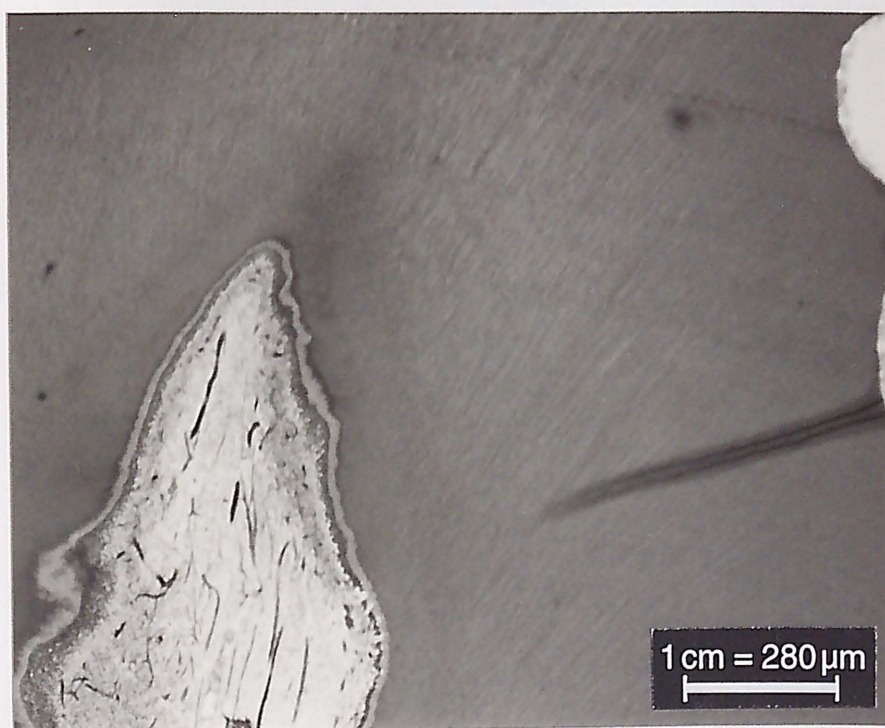


FIGURE 6a. High-speed handpiece preparation. Localized slight disruption of the odontoblastic layer is noted. The dark-staining fold in the dentin is artifact. (Hematoxylin and eosin stain.)

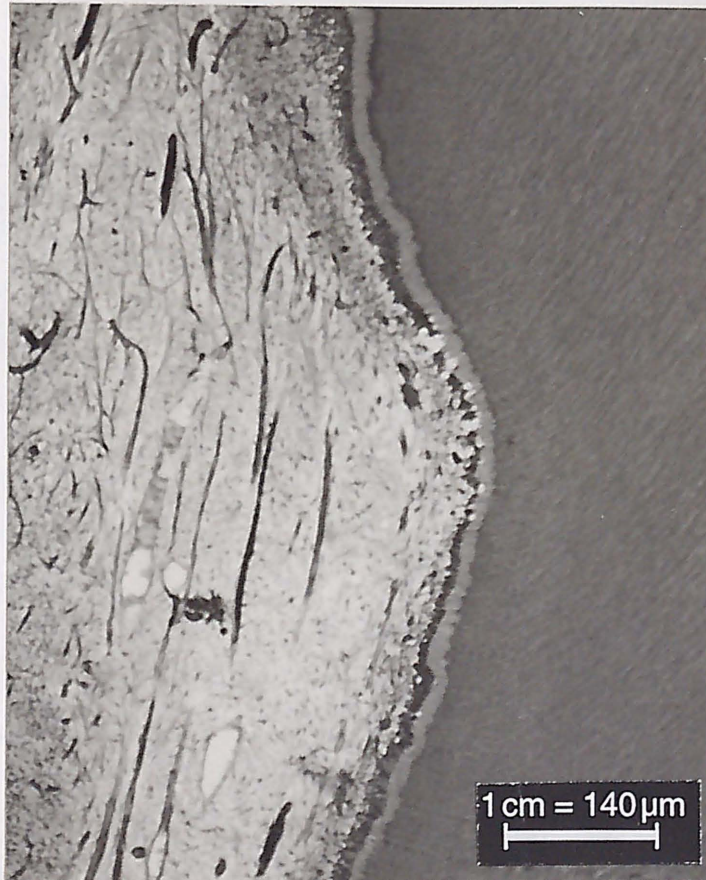


FIGURE 6b. Higher magnification of Figure 6a showing localized slight disruption of the odontoblastic layer. (Hematoxylin and eosin stain.)

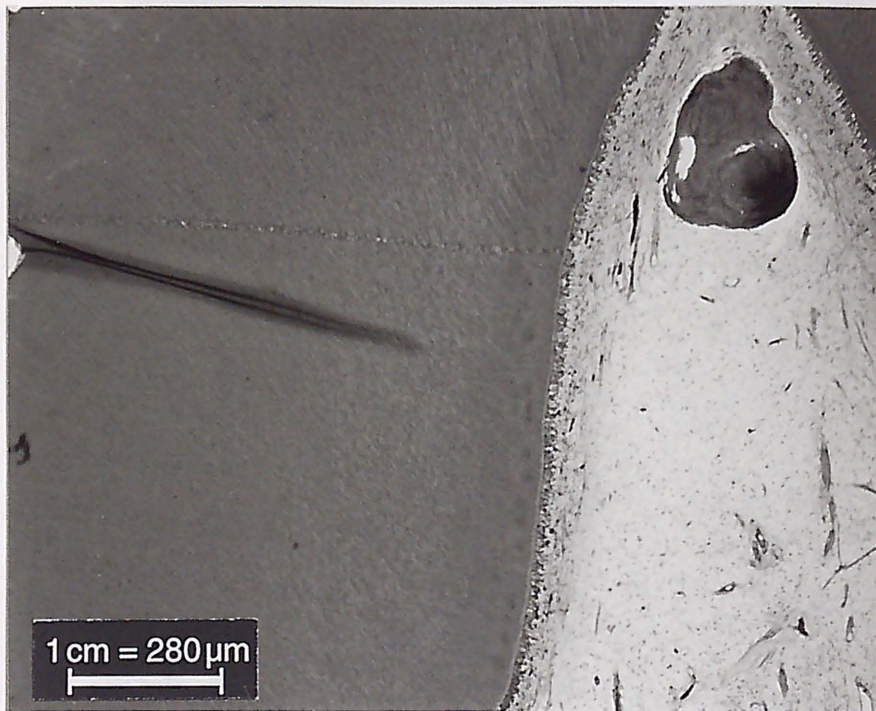


FIGURE 7a. Pulp stone in the coronal portion of the pulp of a tooth that had kinetic cavity preparation, The dark-staining fold in the dentin is artifact. (Hematoxylin and eosin stain.)



FIGURE 7b. Higher magnification of Figure 7a with pulp stone, seen near the top. No pulpal reaction is seen. (Hematoxylin and eosin stain.)



FIGURE 8. Diffuse calcifications of the radicular pulp. This tooth was prepared with the high-speed handpiece. (Hematoxylin and eosin stain.)

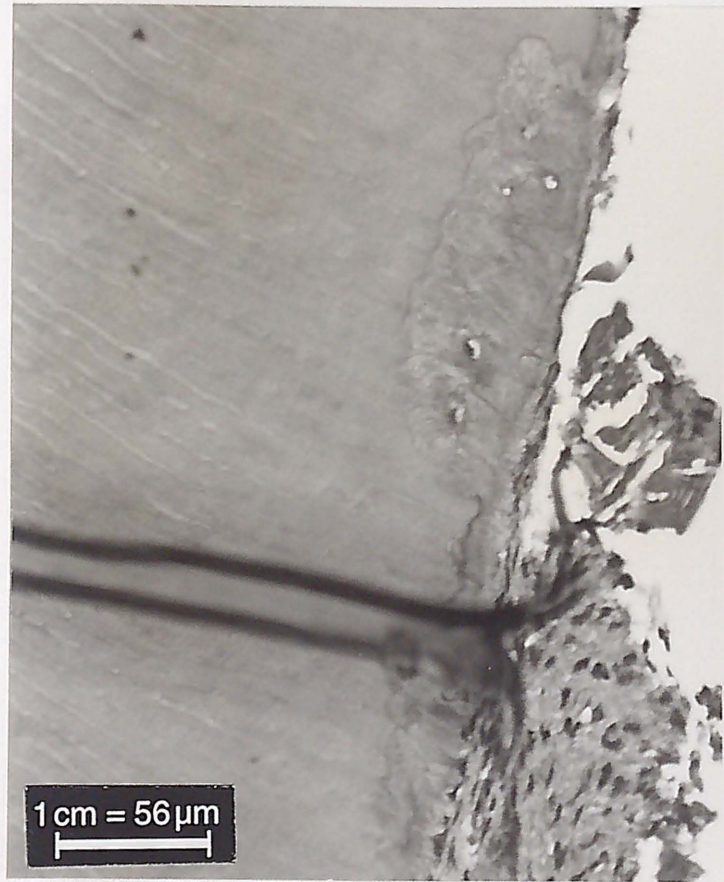


FIGURE 9. External resorption with reversal and repair. Remnants of the periodontal ligament are present. The dark-staining fold in the dentin is artifact. (Hematoxylin and eosin stain.)

TABLE I
Kinetic cavity preparation results

Specimen No.	Remaining Dentin Thickness	Response
1	No preparation	None
4	No preparation	None
5	No preparation	None
7	2021 μm	None
10	2329 μm	None
11	2043 μm	None
13	2034 μm	None
16	2106 μm	None
17	1436 μm	Mild
20	1757 μm	None
21	No preparation	None
24	2216 μm	None
25	1742 μm	None

TABLE II
High-speed handpiece preparation results

Specimen No.	Remaining Dentin Thickness	Response
2	2720 μm	None
3	No preparation	None
6	2099 μm	Mild
8	2158 μm	None
9	1919 μm	None
12	2045 μm	None
14	No preparation	None
15	1548 μm	None
18	1553 μm	None
19	1607 μm	Mild
22	2041 μm	None
23	1823 μm	None
26	1721 μm	Mild

TABLE III

Other histologic findings

Finding	Specimen No.	Preparation	Remaining Dentin Thickness	Pulpal Reaction
Pulp Stone	5	kinetic cavity preparation	no preparation	None
Pulp Stone	7	kinetic cavity preparation	2020 μm	None
Pulp Stone	11	kinetic cavity preparation	2043 μm	None
Pulp Stone	12	high-speed handpiece	2045 μm	None
Diffuse Calcification	22	high-speed handpiece	2040 μm	None
Diffuse Calcification	23	high-speed handpiece	1822 μm	None
Diffuse Calcification	24	kinetic cavity preparation	2216 μm	None
Diffuse Calcification/ External Resorption	21	kinetic cavity preparation	no preparation	None

DISCUSSION

The results demonstrated that in 13 specimens prepared with the air abrasion technique, a mild response was noted in one specimen; no response was noted in 8 specimens, and no preparation into dentin was noted in 4 specimens. In 13 teeth prepared with the high-speed handpiece, 3 showed a mild response; 8 showed no response, and 2 had no preparation into dentin. There was no significant difference in the amount of remaining dentin between the two types of preparation or in the responses observed.

The teeth in this study were prepared to ideal depth in order to simulate the expected clinical use of the air abrasion unit. However, the clinically ideal depth was observed to lack penetration into dentin in several cases and to remove only minimal amounts of dentin in the remaining specimens. The air abrasion technique removed enamel very slowly, even with the use of a different angulation of the nozzle, different tooth-to-nozzle distance, and changes in the speed of movement of the nozzle mesial-distally or occlusal-lingually. The high-speed preparations were also very shallow, which could have been related to the patient population. The age of patients ranged from 9 to 15 years. The eruption of the premolars was not complete in most cases, so that the cemento-enamel junctions were located well below the gingival margin. Preparations were made as close to the gingival level as possible. However, the rubber dam clamp was placed in an atraumatic manner to avoid deep subgingival seating, so that it is likely the gingival levels of the preparations coincided with a significant thickness of enamel in those areas. The preparations with both the handpiece and the air abrasion unit appeared

to be deep, but upon decalcification the depth was found to be only slightly into the dentin, if at all.

The depth of preparation may be the single most important factor in pulpal response. In studies done as early as 1939, Van Huysen and Gurley²³ found that the remaining dentin thickness influenced the severity of pulp reaction. Various studies have reported that a dentin thickness of 2.0 mm or more provides adequate protection to the pulp.^{27-31,54} Seltzer et al.⁵⁶ stated that 1.2 mm of dentin was protective, while Mitchell et al.⁵⁷ related a thickness of 0.4 mm or more to only a mild pulpal reaction. Mjor,⁶⁴ in determining the criteria for study of the biologic effects of restorative materials, advised that preparations must be in the inner third of dentin to produce a meaningful result. The Federation Dentaire International now recommends that the remaining dentin thickness be limited to 1 mm or less in order to test restorative materials.³⁴ With this in mind, little or no pulp response would be expected in the test specimens of the present study due to the ample thickness of remaining dentin.

The lack of pulpal response of the specimens in this study could also be related to the period of time between the preparation and extraction of the teeth. Lisanti and Zander⁴¹ showed a normal histologic appearance of the pulp tissue in dogs one week after application of 300 °F to the pulp. They observed that all histologic changes in the pulp resolve within 2 months, with the extent of the pulp changes and healing times dependent on the amount of insult to the tissues. The conclusion was that the normal dental pulp is able to recover from any harmful effects of normal operative procedures. Swerdlow and Stanley's⁴⁸ examination of the pulp tissue of human teeth also indicated that only a mild

reaction would be expected at 10 to 11 days after tooth preparation when the remaining dentin thickness was 2.0 mm or less. With minimal or no penetration into dentin, pulpal responses in the present study would be expected to be resolved in the 10-to-15-day period that elapsed between cavity preparation and extraction.

In the present study, odontoblastic aspiration, or displacement into tubules, was one of the most common changes in the pulp. This phenomenon was evaluated by Kramer and McLean²⁵ in 1952 in their attempt to establish criteria for the assessment of pulp response. The partial or complete disappearance of the odontoblast at the pulpal end of the cut dentinal tubules was seen. The odontoblast cell bodies could be found at various distances within the dentinal tubule. Swerdlow and Stanley⁴⁸ and James and Schour⁸¹ attributed odontoblastic displacement to edema in the pulp. James and Schour⁸² in another study reported that aspiration of odontoblasts was not due to the cutting procedure but to failure to fill the cavity, so that the pressure within the pulp was greater than within the unfilled cavity. Stanley and Swerdlow⁸³ later determined that the phenomenon of odontoblastic displacement is the result of any circumstance that results in increased intrapulpal pressure, including operative trauma and forceps trauma during extraction. In the present study, because no other histologic signs of trauma were seen in the pulp, dentin or cementum, the presence of odontoblastic displacement could be attributed to the cavity preparation.

Pulp stones, or denticles, are calcified masses that may occur in one or several teeth. They may occur in any age group but are more common in older patients. Pulp stones have been described in young patients, although not frequently.⁸⁴ Diffuse calcification of the pulp is considered to be a true regressive age change.²¹ However, both

pulp stones and diffuse calcifications have been reported to occur in low prevalence in primary teeth.⁸⁸ Diffuse calcifications are composed of irregular calcified material that may be found at the core of most pulp stones and have been surmised to be an early stage of pulp stone formation.⁸⁹ The finding of diffuse calcification in the permanent teeth of a young, healthy patient is unusual and cannot be explained.

External resorption has been investigated for many years⁸⁵ and has been associated with orthodontic tooth movement, as well as with other factors such as trauma and age.⁸⁶ While the apex of the tooth is most commonly affected, other surfaces are also susceptible.⁸⁵ An investigation by Engstrom et al.⁸⁷ found that orthodontic external resorption occurs in reorganizing areas of the periodontal ligament and alveolar bone.

In a future study, preparations made to a depth within 1.0 mm of the pulp and using a larger sample size would be more likely to produce definitive results on the effects of air abrasion and high-speed handpiece preparation on the human dental pulp. A larger sample size will provide the ability to statistically detect any differences in pulpal response.

This study demonstrated that the air abrasion unit can be used for today's recommended purposes without endangering vital pulpal tissues. While the number of teeth with pulpal reaction was greater with high-speed handpiece preparation than with kinetic cavity preparation, this result may be due to the lack of coolant during preparation with the high-speed handpiece. Removal of organic debris in occlusal grooves prior to sealant placement, and freshening of the surface of a fractured resin prior to restoration would not be expected to require deep preparation with the air abrasion unit. The results

of the present study indicate that ideal preparations cause no irreversible damage to the pulpal tissues.

SUMMARY AND CONCLUSIONS

The purpose of this investigation was to compare the histopathologic effects of kinetic cavity preparation to the histopathologic effects of conventional high-speed handpiece preparation on the human dental pulp.

Seven patients who required extraction of teeth for orthodontic purposes were selected. Class V cavity preparations were made in 26 teeth. Thirteen teeth were prepared with kinetic cavity preparation, using 27- μm particles at 160 pounds per square inch pressure. Thirteen contralateral teeth were prepared with the high-speed handpiece and 330 bur. In all teeth, the preparation was conditioned using Ketac Conditioner, rinsed, and air dried. Photac-Fil glass ionomer restorations were placed.

The teeth were extracted between 10 and 15 days after preparation. On teeth with closed apices, the apical one-third of the root was removed. All teeth were placed in 10 percent formalin solution. Serial sections 7- μm thick were cut in the bucco-lingual plane. Selected slides were stained with hematoxylin and eosin, depending on the observation of preparation into the dentin.

Histologic examination indicated that the amount of remaining dentin was of sufficient thickness to protect the pulp from preparation trauma. The mean remaining dentin thickness in teeth prepared using the kinetic cavity preparation was 1965 μm , and in teeth prepared with the high-speed handpiece, 1930 μm . Pulpal responses of all specimens in this study ranged from no response to a mild response. Overall, 22 specimens showed no response, and 4 showed a mild response. In teeth with mild

response, one of the preparations was made with kinetic cavity preparation, and three with high-speed handpiece.

Based on the results of this study, it was concluded that ideal preparation depth into the dentin does not cause irreversible pulpal damage at 10 to 15 days post-preparation, by using either kinetic cavity preparation or high-speed handpiece preparation. The hypothesis that kinetic cavity preparation results in significantly fewer pulpal effects than does conventional high-speed-handpiece preparation is rejected.

THE SOUTH-WEST
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REFERENCES

1. Black RB. Technique for nonmechanical preparation of cavities and prophylaxis. *J Am Dent Assoc* 1945;32:955-65.
2. Black RB. Airbrasive: some fundamentals. *J Am Dent Assoc* 1950;41:701-10.
3. Myers GE. The airbrasive technique. *Br Dent J* 1954;97:291-5.
4. Postle HH., Lefkowitz W. The present status of airbrasive and ultrasonic equipment. *Dent Clin North Am* 1957;1:43-63.
5. Altmann JL. The biophysical basis for new rounded cavity forms. *Int Dent J* 1963;13:582-5.
6. Johnson EW, Castaldi CR, Gau DJ, Wysocki GP. Stress pattern variations in operatively prepared human teeth, studied by three-dimensional photoelasticity. *J Dent Res* 1968;45:548-58.
7. Goldstein RE, Parkins FM. Air-abrasive technology: its new role in restorative dentistry. *J Am Dent Assoc* 1994;125:551-7.
8. Laurell KA, Carpenter W, Daugherty D, Beck M. Histopathologic effects of kinetic cavity preparation for the removal of enamel and dentin. *Oral Surg Oral Med Oral Path* 1995;80:214-25.
9. Gaintantzopoulou MD. Pulpal effects of light-activated glass ionomer lining cements: a histological study. [Thesis]. Indianapolis: Indiana University School of Dentistry, 1990.
10. Black RB. Application and reevaluation of airbrasive technique. *J Am Dent Assoc* 1955;50:408-14.
11. Mann WR. The airdent unit and the airbrasive technique. *J Mich State Dent Soc* 1950;32:23-8.
12. Goldberg MA. Airbrasive: patient reactions. [Abstract]. *J Dent Res* 1952;31:504-5.
13. Morrison AH, Berman, I. Evaluation of the airdent unit: preliminary report. *J Am Dent Assoc* 1953;46:298-303.

14. Epstein S. Analysis of airbrasive procedures in dental practice. *J Am Dent Assoc* 1951;43:578-82.
15. McGehee WHO, True TA, Inskipp EF, eds. A textbook of operative dentistry. 4th ed. New York: McGraw-Hill, 1956:266-73.
16. Fullmer HM, Eastman RF. Effects of aluminum oxide on the rabbit lung. *J Dent Res* 1952;31:487.
17. Kerr DA, Ramfjord S, Ramfjord GM. Effect of inhalation of airbrasive. [Abstract]. *J Dent Res* 1954;33:666.
18. Goldstein RE, Parkins FM. Using air-abrasive technology to diagnose and restore pit and fissure caries. *J Am Dent Assoc* 1995;126:761-6.
19. Burbach G. Micro-invasive cavity preparation with an airbrasive unit. *GP* 1993;2:55-8.
20. Beebe DM. Efficiency of high operating speeds with water lubrication in cavity preparation. *J Am Dent Assoc* 1954;49:650-5.
21. Ten Cate AR. Oral histology: development, structure and function. 3rd ed. St. Louis: CV Mosby, 1989:49, 158-9, 171-86, 395-405.
22. Gurley WB, Van Huysen G. Histologic changes in teeth due to plastic filling materials. *J Am Dent Assoc* 1937;24:1806-16.
23. Van Huysen G, Gurley WB. Histologic changes in teeth of dogs following preparation of cavities of various depths and their exposure to oral fluids. *J Am Dent Assoc* 1939;26:87-100.
24. Van Huysen G, Boyd DA. Operative procedures and the tooth. *J Prosthet Dent* 1953;3:818-26.
25. Kramer IRH, McLean JW. The response of the human pulp to self-polymerising acrylic restorations. *Br Dent J* 1952;92:255-61.
26. James VE, Schour I, Spence JM. Response of human pulp to gutta-percha and cavity preparation. *J Am Dent Assoc* 1954;49:639-49.
27. Swerdlow H, Stanley HR. Reaction of the dental pulp to cavity preparation. (Pt 2). At 150,000 rpm with air-water spray. *J Prosthet Dent* 1959;9:121-31.
28. Stanley HR, Swerdlow H. Reaction of the human pulp to cavity preparation: results produced by eight different grinding techniques. *J Am Dent Assoc* 1959;58:49-60.

29. Stanley HR. Design for a human pulp study (Pt I). *Oral Surg Oral Med Oral Pathol* 1968;25:633-47.
30. Stanley HR. Design for a human pulp study (Pt II). *Oral Surg Oral Med Oral Pathol* 1968;25:756-70.
31. Stanley, HR. Methods and criteria in evaluation of dentin and pulp response. *Int Dent J* 1970;20:507-27.
32. American Dental Association Council on Dental Materials and Devices. Recommended standard practices for biological evaluation of dental materials. *J Am Dent Assoc* 1972;84:382-7.
33. American Dental Association Council on Dental Materials and Devices. American National Standards Institute/American Dental Association Document No. 41 for recommended standard practices for biological evaluation of dental materials. *J Am Dent Assoc* 1979;99:697-8.
34. Federation Dentaire International. Commission on Dental Materials, Instruments, Equipment and Therapeutics. Recommended standard practices for biological evaluation of dental materials. *Int Dent J* 1980;30:140-88.
35. Tobias RS, Browne RM, Plant CG, Ingram DV. Pulpal response to a glass ionomer cement. *Br Dent J* 1978;144:345-50.
36. Pameijer CH, Segal E, Richardson S. Pulpal response to a glass ionomer cement in primates. *J Prosthet Dent* 1981;46:36-40.
37. Dogon IL, Vanleeuwen MJ, Heeley JDA. Histological evaluation of a light-cured glass ionomer liner/base. [Abstract]. *J Dent Res* 1989;68:244.
38. Tobias RS, Plant CG, Rippin JW, Browne RM. Pulpal response to an anhydrous glass ionomer luting cement. *Endod Dent Traumatol* 1989;5:242-52.
39. Tobias RS, Browne RM, Plant CG, Williams JA, Rippin JW. Pulpal response to two semihydrous glass ionomer luting cements. *Int Endod J* 1991;24:95-107.
40. Vaughn RC, Peyton FA. The influence of rotational speed in temperature rise during cavity preparation. *J Dent Res* 1951;30:737-44.
41. Lisanti VF, Zander HA. Thermal injury to normal dog teeth: in vivo measurements of pulp temperature increases and their effect on pulp tissue. *J Dent Res* 1952;31:548-58.

42. Lisanti VF, Zander HA. Thermal conductivity of dentin. *J Dent Res* 1950;29:493-7.
43. Peyton FA, Henry E. Problems of cavity preparation with modern instruments. *New York Dent J* 1952;22:147.
44. Peyton FA, Henry EE. The effect of high speed burs, diamond instruments and air abrasive in cutting tooth tissue. *J Am Dent Assoc* 1954;49:426-35.
45. Peyton FA. Temperature rise in teeth developed by rotating instruments. *J Am Dent Assoc* 1955;50:629-32.
46. Peyton FA. Evaluation of dental handpieces for high speed operations. *J Am Dent Assoc* 1955;50:383-91.
47. Peyton FA. Effectiveness of water coolants with rotary cutting instruments. *J Am Dent Assoc* 1958;56:664-75.
48. Swerdlow H, Stanley HR. Reaction of the human dental pulp to cavity preparation. (Pt 1). Effect of water spray at 20,000 rpm. *J Am Dent Assoc* 1958;56:317-29.
49. Marsland EA, Shovelton DS. The effect of cavity preparation on the human dental pulp. *Br Dent J* 1957;102:213-22.
50. Jeserich PH. Factors necessary to minimize thermal changes in tooth structures from operative procedures. [Abstract]. *New York J Dent* 1935;5:275.
51. Stanley HR, Swerdlow H. Biologic effects of various cutting methods in cavity preparation: the part pressure plays in pulpal response. *J Am Dent Assoc* 1960;61:450-6.
52. Lefkowitz W, Robinson HBG, and Postle HH. Pulp response to cavity preparation. *J Prosthet Dent* 1958;8:315-24.
53. Peyton FA, Vaughn RC. Thermal changes developed during the cutting of tooth tissue. *Fortnightly Review Chi D Soc* 1950;20:9-23.
54. Shroff, FR. Effects of filling materials on the dental pulp. *NZ Dent J* 1946;42:99-114, 145-64 and 1947;43:35-58.
55. James VE, Schour I. Early dentinal and pulpal changes following cavity preparation and filling materials in dogs. *Oral Surg Oral Med Oral Pathol* 1955;8:1305-14.

56. Seltzer S, Bender IB, Kaufman IJ. Histologic changes in dental pulps of dogs and monkeys following application of pressure, drugs and microorganisms on prepared cavities. *Oral Surg Oral Med Oral Pathol* 1961;14:327-46.
57. Mitchell DF, Buonocore MG, Shazer S. Pulp reaction to silicate cement and other materials: relation to cavity depth. *J Dent Res* 1962;41:591-5.
58. Dowden, WE. Discussion of: methods and criteria in evaluation of dentin and pulpal responses. *Int Dent J* 1970;20:531-2.
59. Dickey DM, El-Kafrawy AH, Mitchell DF. Clinical and microscopic pulp response to a composite restorative material. *J Am Dent Assoc* 1974;88:108-13.
60. Plant CG, Anderson RJ. The effect of cavity depth on the pulpal response to restorative materials. *Br Dent J* 1978;144:10-13.
61. Plant CG, Knibbs PJ, Tobias RS, Britton AS, Rippin JW. Pulpal response to a glass-ionomer luting cement. *Br Dent J* 1988;165:54-8.
62. Plamondon, TJ, Walton R, Graham GS, Houston G, Snell G. Pulp response to the combined effects of cavity preparation and periodontal ligament injection. *Oper Dent* 1990;15:86-93.
63. Lee S, Walton RE, Osborne JW. Pulp response to bases and cavity depths. *Am J Dent* 1992;5:64-8.
64. Mjor, IA. Usage test for restorative materials. *J Endod* 1978;4:308-11.
65. Wilson AD, Kent BE. A new translucent cement for dentistry. *Br Dent J* 1972;132:133-5.
66. Klotzer WT. Pulp reactions to a glass ionomer cement. [Abstract]. *J Dent Res* 1975;54:678.
67. Dahl BL, Tronstad L. Biological tests of an experimental glass ionomer (silicopolyacrylate) cement. *J Oral Rehabil* 1976;3:19-24.
68. Nordenvall K, Brannstrom M, Torstensson B. Pulp reactions and microorganisms under ASPA and Concise composite fillings. *J Dent Child* 1979;46:449-53.
69. Kawahara H, Imanishi Y, Oshima H. Biological evaluation of glass ionomer cement. *J Dent Res* 1979;58:1080-6.
70. Cooper IR. The response of the human dental pulp to glass ionomer cements. *Int Endod J* 1980;13:76-88.

71. Heys RJ, Fitzgerald M, Heys DR, Charbeneau GT. An evaluation of a glass ionomer luting agent: pulpal histological response. *J Am Dent Assoc* 1987;114:607-11.
72. Paterson RC, Watts A. The response of the rat molar pulp to a glass ionomer cement. *Br Dent J* 1981;151:228-30.
73. Pameijer CH, Stanley HR. Primate pulp response to anhydrous Chembond. [Abstract]. *J Dent Res* 1984;63:171.
74. Ucok M. Biological evaluation of glass ionomer cements. *Int Endod J* 1986;19:285-97.
75. Felton DA, Cox CF, Odom M. Histologic study of a light cured glass ionomer cavity liner. [Abstract]. *J Dent Res* 1987;67:302.
76. Paterson RC, Watts A. Toxicity to the pulp of a glass-ionomer cement. *Br Dent J* 1987;162:110-2.
77. Mjor IA, Nordahl I, Tronstad L. Glass ionomer cements and dental pulp. *Endod Dent Traumatol* 1991;7:59-64.
78. Felton DA, Cox CF, Odom M, Kanoy BE. Pulpal response to chemically cured and experimental light-cured glass ionomer cavity liners. *J Prosthet Dent* 1991;65:704-12.
79. Stanley HR. Local and systemic responses to dental composites and glass ionomers. *Adv Dent Res* 1992;6:55-64.
80. Luna, LG, ed. Manual of histologic staining methods of the U.S. Armed Forces Institute of Pathology. 3rd ed. New York: McGraw Hill Book Co., 1968:60-1.
81. James VE, Schour I. The effects of cavity preparation and zinc oxide and eugenol upon the human pulp. [Abstract]. *J Dent Res* 1955;34:698.
82. James VE, Schour I. Effect of cavity preparation alone on the human pulp. [Abstract]. *J Dent Res* 1955;34:798.
83. Stanley HR, Swerdlow H. Aspiration of cells into dentinal tubules? *Oral Surg Oral Med Oral Pathol* 1958;11:1007-17.
84. Siskos GJ, Georgopoulou M. Unusual case of general pulp calcification (pulp stones) in a young Greek girl. *Endod Dent Traumatol* 1990;6:282-4.
85. Henry JL, Weinmann JP. The pattern of resorption and repair of cementum. *J Am Dent Assoc* 1951;42:270-90.

86. Brezniak N, Wasserstein A. Root resorption after orthodontic treatment (Pts 1 & 2). *Am J Orthod Dentofac Orthop* 1993;103:62-6, 138-46.
87. Engstrom C, Granstrom C, Thilander B. Effect of orthodontic force on periodontal tissue metabolism. *Am J Orthod Dentofac Orthop* 1988;93:486-95.
88. Kumar S, Mathus RM, Chandra S, Jaiswal JN. Pulp calcifications in primary teeth. *J Pedod* 1990;14:93-6.
89. Moss-Salentijn L, Hendricks-Klyvert M. Calcified structures in human dental pulps. *J Endod* 1988;14:184-9.

ABSTRACT

EFFECTS OF KINETIC CAVITY PREPARATION VS.
CONVENTIONAL HANDPIECE PREPARATION
ON THE HUMAN DENTAL PULP

by

Julie M. Collins

Indiana University School of Dentistry
Indianapolis, Indiana

The purpose of this investigation was to compare the histopathologic effects of kinetic cavity preparation to the histopathologic effects of conventional high-speed handpiece preparation on the human dental pulp. The objective was to test the following hypothesis: kinetic cavity preparation results in significantly fewer pulpal effects than does conventional preparation using the high-speed handpiece.

Class V cavity preparations were made in 26 teeth of seven patients who required extraction of these teeth for orthodontic purposes. Thirteen teeth were prepared using kinetic cavity preparation, using 27- μ m aluminum oxide particles at 160 pounds per square inch pressure. Thirteen were prepared using the high-speed handpiece and 330 bur. Glass ionomer restorations were placed in all teeth. Extractions were done 10 to 15

days after preparation. On teeth with closed apices, the apical one-third of the root was removed. All teeth were placed in 10 percent formalin solution. Teeth were sectioned and selected slides stained with hematoxylin and eosin for histologic evaluation.

Microscopic findings indicated that the amount of remaining dentin was of significant thickness to be protective to the pulp. Pulpal responses ranged from no response in 22 specimens to a mild response in 4 specimens.

Based on the results of this study, it was concluded that shallow preparation into the dentin does not cause pulpal damage at 10 to 15 days post-preparation, when using either kinetic cavity preparation or high-speed handpiece preparation. The hypothesis that kinetic cavity preparation causes significantly fewer pulpal effects than does conventional preparation with the high-speed handpiece was rejected.

CURRICULUM VITAE

JULIE M. COLLINS

September 19, 1962	Born in Chicago, Illinois
1980 to 1984	B.A., Chemistry Monmouth College Monmouth, Illinois
1984 to 1988	D.D.S. University of Illinois at Chicago College of Dentistry Chicago, Illinois
1988 to present	Commissioned in the United States Air Force
1988 to 1989	General Practice Residency Sheppard Air Force Base, Texas
1989 to 1996	General Dental Officer RAF Bentwaters, England Whiteman AFB, Missouri
1996 to 1998	Certificate in Pediatric Dentistry James Whitcomb Riley Hospital for Children and Indiana University School of Dentistry Indianapolis, Indiana

Professional Organizations

American Academy of Pediatric Dentistry
American Dental Association
American Society of Dentistry for Children